Immunology

Assisted suicide for B cells

J. Exp. Med. **197,** 1125–1139 (2003)

Infectious organisms have a variety of ways of protecting themselves from the immune systems of their unwitting hosts. Carl S. Goodyear and Gregg J. Silverman now show that, for the bacterium *Staphylococcus aureus*, attack is the best form of defence.

When the immune system's B cells encounter unfamiliar items, such as material from a bacterium, they normally respond by differentiating into cells that produce antibodies against the pathogen. They also generate long-lasting memory B cells, which deal rapidly with the intruder if reinfection occurs.

Goodyear and Silverman find that these events do not take place when mice are exposed to a protein called SpA, produced by *S. aureus*. The protein binds to B cells and causes them to commit suicide within a few hours. Memory B cells never have the chance to develop. The authors believe that these processes also occur during natural *S. aureus* infections.

The findings could aid the development of vaccines against *S. aureus*. But what Silverman and colleagues are currently working on is a treatment for systemic lupus erythematosus, an autoimmune disease caused by faulty B cells. The idea is to use the bacterial SpA protein to kill the subset of B cells that cause the disease. **Lisen Arnheim**

Microfluidics

Traffic flow

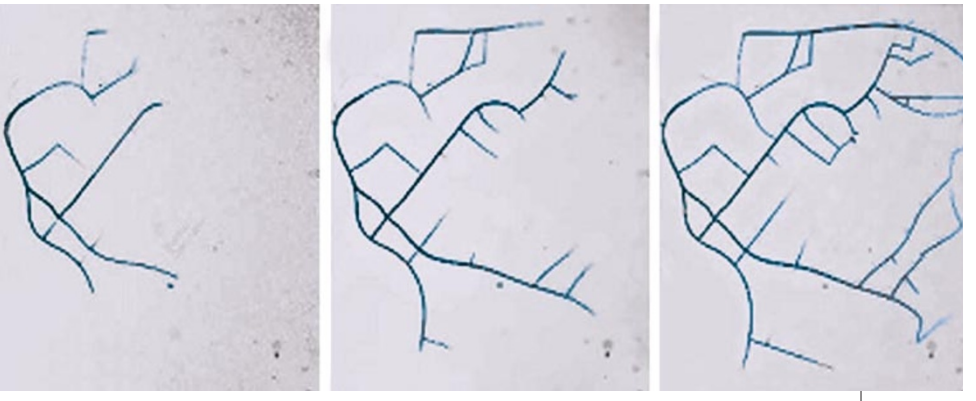
Langmuir doi:10.1021/la030054x (2003)

What's the fastest route across downtown Boston? Michael J. Fuerstman and colleagues can tell you, by using a microfluidic system to find the way through the complex maze of the city and identify the best path.

Fuerstman *et al.* have built a number of mazes — including one representing a street plan of Boston — from networks of microchannels imprinted in poly(dimethylsiloxane), sealed with a flat surface. The mazes are filled with one fluid, then a contrasting dye solution is introduced and its movements traced as it explores, under an applied pressure gradient, all continuous paths between inlet and outlet.

The inlet of one such system (pictured) marks the site of the Boston Museum of Science, and is connected to the outlet — the Computer Museum — through channels representing some of the city's streets and highways. The different widths of the microfluidic channels control the velocity of the fluid to roughly match the relative driving speeds on the corresponding roads. Introducing a blue liquid into the pre-filled network reveals two routes between the museums, one slightly faster than the other.

In a maze possessing multiple solutions, the tracing fluid reaches the outlet fastest along the path of least fluidic resistance. Adjusting the properties of the channels and fluids will affect the compromise found between the shortest and widest channels. The approach, suggest the authors, might be used to model complex nonlinear problems such as traffic flow or distribution systems. **Magdalena Helmer**



Which way? Dye running through a microfluidic representation of the streets of Boston signals the quickest route across town. These images were taken over a five-second interval.

Cancer

Brake for cell division

Genes Dev. **17,** 1201–1206 (2003)

The *tob* gene is one of many that prevent cells from dividing endlessly. Yutaka Yoshida and colleagues now provide evidence that *tob* also suppresses tumour formation.

It was already known that cells without Tob protein continue to multiply under circumstances where other cells give up — during famine, for instance. But it was not clear whether Tob is connected with the uncontrolled cell growth that leads to tumour formation.

Enter Yoshida *et al.*, who have generated *tob*less mice. The authors find that tumours grow spontaneously in various tissues in these mice, including the lymph nodes and lungs. In addition, more *tob*-deficient animals than control mice develop liver tumours, especially after treatment with a liver-specific cancer-causing compound. This is of interest as there are currently no adequate animal models for liver cancer.

Yoshida *et al.* further show that Tob can repress the expression of cyclin D1, which drives cell proliferation. So, when Tob activity is reduced, cyclin D1 levels rise, and cell division goes into overdrive. Although analysis of more than 50 human tumours did not reveal any mutations in the *tob* gene, its expression level was reduced in several human lung tumours. So it seems that suppression of Tob expression may contribute to tumour progression. **Marie-Thérèse Heemels**

Biomaterials

Size matters

Proc. Natl Acad. Sci. USA **100,** 5597–5600 (2003)

Biological composite materials such as bone, shell and tooth are renowned for their strength. They generally consist of crystals or platelets of a hard mineral, often calcium carbonate, embedded in a soft matrix of proteins and other organic molecules. Huajian Gao and colleagues say that the size of the mineral particles — typically tens to hundreds of nanometres — is not arbitrary. Rather, it seems to be tuned to give the mineral a remarkable characteristic: it is insensitive to the presence of structural flaws.

In hard, brittle materials, flaws such as microscopic scratches or holes are an Achilles' heel, with a disruptive potential that belies their size. Stresses in the solid become concentrated at these places, nucleating cracks that travel rapidly through the material. But the analysis of Gao *et al.* shows that the fracture resistance of a plate containing a surface flaw increases as the plate thickness decreases, becoming equal to that of a perfect, unflawed plate at a thickness of around 30 nm. The exact value depends on the precise properties of the material, but it seems clear that the weakening effect of flaws vanishes specifically at the nanoscale.

The structure of these biomineral particles is also probably governed by constraints on, for example, their nucleation and growth rates. But it seems that attempts to recreate the superior properties of biomaterials in synthetic composites will require mimicry not just of the arrangements of the constituent materials but also of their scale. **Philip Ball**

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